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RESEARCH MODELS :: HYBRID MICE

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Inbred Rats
Outbred Mice
Inbred Mice
Hybrid Mice
- B6C3F1 Mice
- BDF1 Mice
- CDF1 Mice
- CB6F1 Mice

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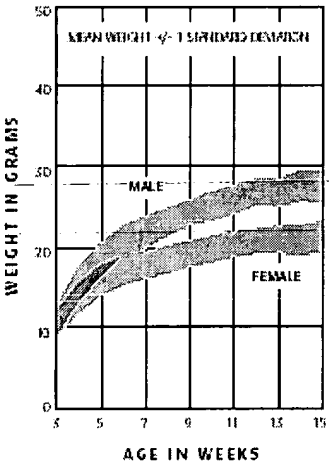
Transgenic Models
Outbred Hamsters/ Guinea Pigs/Gerbils/ Rabbits
Consomic Rats
Disease Models
Surgical Services
SPF Eggs

CDF1 Mice

	MALE		FEMALE	
Weight in Grams	Approximate Age in Days	Price	Approximate Age in Days	Price
Up to 12	*Up to 23*	10.95	*Up to 23*	12.00
13-15	24-27	12.65	24-29	13.60
16-18	28-32	13.50	30-44	14.55
19-21	33-42	14.35	45-70	15.65
22-24	43-63	16.60	71-91	17.90
25 *plus*	*Prices upon request*		*Prices upon request*	
Littermates *21 days old only*		18.05		18.05

NOMENCLATURE CD2F1/CrlBR **ORIGIN** A cross between female BALB/cAnNCrlBR x male DBA/2NCrlBR. **COAT COLOR** Brown.



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Cell Lines

ATCC Number: CCL-219

Order this item

Price: $215.00

Designation: L1210

Depositors: EM Jensen

Biosafety Level: 1

Shipped: frozen

Medium & Serum: See Propagation

Growth Properties: suspension

Organism: *Mus musculus* (mouse)

Morphology: lymphoblast

Tissue: lymphocytic leukemia

Permits/Forms: In addition to the MTA mentioned above, other ATCC and/or regulatory permits may be required for the transfer of this ATCC material. Anyone purchasing ATCC material is ultimately responsible for obtaining the permits. Please click here for information regarding the specific requirements for shipment to your location.

Related Cell Culture Products

Comments:	Tested and found negative for ectromelia virus (mousepox). The line was established from a tumor in female #234 that developed following skin paintings with 0.2% methylcholanthrene in ether. [22513]
Tumorigenic:	yes, Tumors developed within 21 days at 100% frequency (5/5) in nude mice inoculated subcutaneously with 10(7) cells.
Reverse Transcript:	positive
Strain:	DBA subline 212
Age Stage:	8 months
Gender:	from female organisms(s)
Passage submitted to the ATCC:	377
Propagati n:	ATCC medium: Dulbecco's modified Eagle's medium with 4.5 g/L glucose, 90%; horse

	serum, 10% - OR - Fischer's medium, 90%; horse serum, 10%
Subculturing:	Cultures can be maintained by addition or replacement of fresh medium. Start cultures at 5 X 10 exp4 viable cells/ml.
Fluid Renewal:	Add fresh medium (20% to 30% by volume) every 2 to 4 days
D ubling Time:	8 to 10 hrs
References:	22513: J. Natl. Cancer Inst. 10: 179-192, 1949. 26055: J. Natl. Cancer Inst. 36: 405-421, 1966. 26056: Cancer Chemother. Rep. 51: 451-453, 1967. 32923: Young SW , et al. Gadolinium(III) texaphyrin: a tumor selective radiation sensitizer that is detectable by MRI. Proc. Natl. Acad. Sci. USA 93: 6610-6615, 1996. PubMed: 8692865

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☐ 1: Blood 1990 Oct 1;76(7):1308-14 Related Articles, Link

Direct stimulation of cells expressing receptors for macrophage colony-stimulating factor (CSF-1) by a plasma membrane-bound precursor of human CSF-1.

Stein J, Borzillo GV, Rettenmier CW.

Department of Hematology/Oncology, St Jude Children's Research Hospital, Memphis, TN.

Secreted forms of macrophage colony-stimulating factor (M-CSF or CSF-1) are generated by proteolytic cleavage of membrane-bound glycoprotein precursors. Alternatively spliced transcripts of the human CSF-1 gene encod at least two different transmembrane precursors that are differentially processed in mammalian expression systems. The larger precursor rapidly undergoes proteolysis to yield the secreted growth factor and does not give rise to forms of CSF-1 detected on the cell surface. By contrast, the smaller human CSF-1 precursor is stably expressed on the plasma membrane where it is inefficiently cleaved to release a soluble molecule. To determine whethe the smaller precursor is biologically active on the cell surface, mouse NIH-3T3 fibroblasts expressing the different forms of human CSF-1 were killed by chemical fixation and tested for their ability to support the proliferation of cells that require this growth factor. Only fixed cells expressing human CSF-1 precursors on their surface stimulated the growth in vitro of a murine macrophage cell line or normal mouse bone marrow-derived mononuclear phagocytes. The ability of these nonviable fibroblasts to induce the proliferation of CSF-1-dependent cells was not mediated by release of soluble growth factor, required direct contact with the target cells, and was blocked by neutralizing antiserum to CSF-1. These results demonstrate that the cell surface form of the human CSF-1 precursor is biologically active and indicate that plasma membrane-bound growth factors can functionally interact with receptor-bearing targets by direct cell-cell contact.

PMID: 2145044 [PubMed - indexed for MEDLINE]

1: Dev Comp Immunol 1995 May-Jun;19(3):261-77 Related Articles, Link

ELSEVIER SCIENCE FULL-TEXT ARTICLE

The identification of the neonatal NBXFO hybridoma cell and its mediator.

Jadus MR, Irwin MR, Barton BE, Irwin MC, Wepsic HT.

Department of Laboratory Service, Veterans Affairs Medical Center, Long Beach, CA 90822, USA.

The neonatal spleen:myeloma hybridoma cell, NBXFO, with immunosuppressive properties supported rodent hematopoietic colony formation. We identified this hybridoma to be an undifferentiated fibroblast that produced macrophage colony-stimulating factor (M-CSF). The bone marrow cells that grew in the presence of the NBXFO supernate were macrophages and were immunosuppressive towards lymphocytes. Neutralizing anti-M-CSF antibody partially inhibited the actions of the neonatal splenic suppressor cells. Neonatal splenocytes, but not the other parental cell line, FO, induced macrophage colony formation, possessed surface-associated M-CSF, and possessed M-CSF-specific transcripts. Therefore, we believe that the M-CSF-producing phenotype was contributed by a fibroblastic stromal cell and that these stromal cells could be responsible for the in situ generation of neonatal splenic suppressor cells.

PMID: 8595824 [PubMed - indexed for MEDLINE]

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Doubling Time:	8 to 10 hrs
References:	22513: J. Natl. Cancer Inst. 10: 179-192, 1949. 26055: J. Natl. Cancer Inst. 36: 405-421, 1966. 26056: Cancer Chemother. Rep. 51: 451-453, 1967. 32923: Young SW , et al. Gadolinium(III) texaphyrin: a tumor selective radiation sensitizer that is detectable by MRI. Proc. Natl. Acad. Sci. USA 93: 6610-6615, 1996. PubMed: 8692865

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1: J Natl Cancer Inst 1995 Jun 7;87(11):809-16 Related Articles, Link

Comment in:
- J Natl Cancer Inst. 1995 Nov 15;87(22):1716-7.

Macrophage colony-stimulating factor complementary DNA: a candidate for gene therapy in metastatic melanoma.

Walsh P, Dorner A, Duke RC, Su LJ, Glode LM.

(Department of Dermatology), University of Colorado Cancer Center, University of Colorado Health Sciences Center, Denver 80262, USA.

BACKGROUND: At present, there is no highly effective treatment for metastatic melanoma. Innovative approaches aimed at inducing a more effective immune response against tumors have shown promising results in animal models. One approach involves the genetic modification of tumor cells so that they produce cytokines that stimulate an immune response. PURPOSE: The aim of this study was to determine the effectiveness of cytokine gene therapy for metastatic melanoma in a murine melanoma model. METHODS: B16F10 murine melanoma cells, which readily metastasize to the lungs, were transduced with a retroviral vector containing genes encoding neomycin resistance and human macrophage colony-stimulating factor (M-CSF). The presence of M-CSF messenger RNA in transduced cells was examined by coupled reverse transcription and polymerase chain reaction. Concentrations of soluble M-CSF in cell culture supernatants were determined by enzyme-linked immunosorbent assays (ELISAs). A clonal cell line, designated N+/CSF+, that expressed and secreted M-CSF was identified. Another clonal cell line, designated N+/CSF , did not secrete M-CSF at levels detectable by ELISA. B16F10, N+/CSF-, and N+/CSF+ cells, individually or in combination, were injected intravenously or subcutaneously into C57BL/6 mice; we then evaluated the tumorigenicity and metastatic behavior of the cells, as well as the immune responses and survival of the mice. The immune responses assayed were the cytotoxic T lymphocyte (CTL) and peritoneal exudate cell (PEC) tumoricida activities. RESULTS: Injection of B16F10 cells into the tail vein of C57BL/6 mice led to the establishment of lung metastases by week 2 and death by week 8. Injection of the N+/CSF+ or N+/CSF- cells led to the establishment of lung metastases that were detected at 2 and 3 weeks, respectively; however, these metastatic lesions were eliminated, and the animals had

survival rates similar to those of the noninjected control mice. Injection of mice with a mixture of B16F10 and N+/CSF- cells resulted in the development of metastatic disease and 0% survival at 8 weeks, whereas mice that had been given an injection of a mixture of B16F10 and N+/CSF+ cells had an 80% survival rate at 8 weeks and survived at least two times longer (P = .007). The CTL and PEC tumoricidal activities in animals given an injection of N+/CSF+ cells suggested that monocytes and lymphocytes were responsible for the observed antitumor response. CONCLUSION: These findings suggest that the expression of M-CSF by genetically modified melanoma cells caused an effective antitumor immune response in host C57BL/6 mice and, thus, prolonged survival over that observed in the contro mice.

PMID: 7791230 [PubMed - indexed for MEDLINE]

☐ 1: Eur J Immunol 1993 Jan;23(1):186-90 Related Articles, Link

Macrophage colony-stimulating factor gene transfer into tumor cells induces macrophage infiltration but not tumor suppression.

Dorsch M, Hock H, Kunzendorf U, Diamantstein T, Blankenstein T.

Institut fur Immunologie, Universitatsklinikum Steglitz, Freie Universitat Berlin, FRG.

In order to analyze the effect of a high local concentration of macrophage colony-stimulating factor (M-CSF; CSF-1) on tumor growth, the plasmacytoma cell line J558L was transfected with the human M-CSF gene and injected into syngeneic BALB/c mice. In contrast to the parental tumors, M-CSF transfectants were heavily infiltrated by macrophages as evidenced by immunohistochemistry with antibodies to Mac-1 and Mac-3 and by isolation of the macrophages from the tumor. Nevertheless, tumor growth was only slightly affected by M-CSF and M-CSF-producing cells grew as tumor in all cases. The growth retardation of M-CSF-producing cells varied depending on the experiment and seemed to be due to an indirect effect because the growth rate of the cells in vitro had not changed upon gene transfer. Attempts to activate the tumor-infiltrating macrophages for tumor suppression by systemic application of interferon-gamma and/or lipopolysaccharide were not successful. Altogether, our results suggest that M-CSF is a potent chemoattractant for macrophages in vivo but alone is not sufficient to activate these macrophages for tumoricidal activity.

PMID: 8419170 [PubMed - indexed for MEDLINE]